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DISEASE CARRIERS[1]

THE past two decades have witnessed many notable achievements in medicine, chief among which, as regards the infectious diseases, stand the discovery of the curative sera, and the more recent discovery of specific chemical agents for the treatment and cure of disease. The real, ultimate goal toward which the research work bearing on human and animal infections points is, and must be, the cure of the afflicted. No royal road leads to the desired end, but instead numberless trails must be blazed which too often lead seemingly to nowhere. While the crowning achievement, the direct conquest of disease, is the aim, the investigator from the beginning has endeavored to accomplish essentially the same result by preventive means. The search for the cause of disease, the recognition of the portals of entry and exit, the perfecting of methods of disinfection, and the development of preventive inoculation served to build up a fairly effective basis for prophylaxis. These methods would, indeed, have sufficed had the earlier views regarding the spread of disease been correct. The general knowledge regarding the highly contagious diseases made it seem probable that all infections were spread, more or less directly, from the sick to the healthy, and as a result preventive measures were applied to the patient and to his immediate surroundings. The outcome, however, was not always satisfactory and the reason is not difficult to see. Fully as

[1] Address of the vice-president and chairman of Section K, American Association for the Advancement of Science, December 29, 1911.

important as a knowledge of the germ itself is the fundamental fact that disease may be conveyed through the agency of apparently healthy insects, lower animals, and even man himself. It is the recognition of this fact, the existence of *disease carriers,* which has brought about such remarkable results in the fight against malaria, yellow fever, Malta fever and many other diseases. Hence a brief outline of our knowledge of this interesting subject may not be out of place.

It may be well at the outset to state that the term "disease carrier" is applied to animals or persons who, though apparently in perfect health, harbor and eliminate a given disease germ. The fact that the "carrier" is an apparently healthy animal means that the disease may be spread through a wholly unexpected source. The old view of the transmission of disease by contact with the sick remains true, but it is enlarged and supplemented by newer facts. Since more or less direct contact with the apparently healthy carrier, or with the actually sick serves to spread an infection, it is clear that preventive measures must consider the former as well as the latter.

Strange as it may seem, the existence of certain carriers, though not their import, was recognized in the early days of bacteriology. Thus, the presence of the microbe of sputum septicemia in the mouths of healthy persons was noted independently by Sternberg and by Pasteur, in 1880, but it was not until several years later that this organism was shown to be the cause of lobar pneumonia. The frequent persistence of this organism in the sputum after recovery was observed at an early date.

An even more striking example was furnished with the discovery of the diphtheria bacillus in 1883, for Loeffler not only found this organism in the sick, but also in some perfectly healthy children. So contradictory to the accepted order of things was this fact that for several years it prevented the full recognition of this germ as the cause of the disease. The more thoroughly this disease was studied, the more it became evident that recovery did not mean an immediate disappearance of the pathogenic microbe. In other words, clinical recovery did not assure freedom from danger to others. The early recognition of this fact led to the establishment of the bacteriologic control of the recovered patient in the form as it now exists in practise. Two or more consecutive examinations of the throat and nose must yield negative findings before it can be said that the danger of spreading the infection has been overcome.

The presence of the pathogenic agent in the active stage of the disease is a necessary condition; the persistence of such agent during convalescence is more or less to be expected, but the continued existence, at times, of the organism in the individual after complete recovery, and its presence in persons who apparently have never had the disease is somewhat difficult of explanation. The three types thus alluded to are conveniently designated as (1) *convalescent,* (2) *chronic* and (3) *healthy carriers.* The latter in the majority of cases perhaps includes individuals who, at some time, and unknown to them, have had an attack of the disease; they are therefore virtually chronic carriers. Others, though healthy, may be in the incubation stage and hence develop the disease some days later. Lastly, a third group would include strictly healthy carriers, those who have never had the disease and are only remotely liable to it. Obviously, then, it is difficult to distinguish between these several groups of healthy carriers.

DIPHTHERIA

As regards diphtheria, investigations have shown that the healthy carriers are never found in regions free from the disease. They are met with only in places where the disease prevails; the home, hospital, asylum, school and, naturally, the large city furnish examples, though in extremely variable number. Intimate association with the patient, either before or during sickness, as is likely to occur in the home, yields the largest percentage of such carriers. This is especially true where preventive measures, such as isolation and disinfection, are not enforced at the outset. Under such conditions it has been found that fully one third of the exposed become carriers for a greater or less length of time. Where proper measures are instituted early, the number is necessarily greatly reduced. Systematic examinations made in the large cities have, time and again, demonstrated the presence of healthy carriers in from 2 to 4 per cent. or more of the persons tested.

The persistence of the organism in the healthy carrier is usually, and fortunately, of short duration, though exceptionally it may continue to be present for months. Such carriers are unquestionably a source of danger to others and serve to explain outbreaks of the disease where contact with a sick person is positively excluded. The fact that the blood of a majority of the healthy carriers possesses antitoxic properties indicates clearly that they have passed through a previous, though unrecognized, mild infection and consequently that they really belong to the type of chronic carriers. A small number of the healthy carriers may eventually themselves develop the disease, while others, for some unknown reason, escape infection.

The convalescent and chronic carriers are properly looked upon as being dangerous to the community. It is a well-recognized fact that the diphtheria bacillus persists in the throat and nose, for a variable period of time, after the disappearance of all clinical symptoms of the disease. It may remain present for a few days, weeks or months. Fortunately, the vast majority rid themselves of the invading organism in a relatively short time, but as long as they harbor the organism they are in a position to infect others. Isolation, under such conditions, is just as necessary as in the acute stage of the infection. The really chronic carrier, the one who harbors the germs for months, if not years, presents the most difficult problem.

TYPHOID FEVER

The studies on typhoid fever during the past few years have been especially fruitful in enlarging our knowledge of human carriers and have served to concentrate attention to the important part played by these in the propagation of the disease. The accepted cause, the typhoid bacillus, is known to be present not only in the intestines, but also in the internal organs and in the blood. Because of the latter occurrence the majority of typhoid patients, after about the twelfth day of the disease, eliminate the typhoid bacillus in the urine, at times in enormous numbers, and such elimination may continue for weeks after complete recovery has taken place. Exceptionally, the typhoid bacillus may persist in the urine or discharges for months, and years, if not through the remainder of life.

The problem of the healthy carrier is one of special import. Here, as before, we have those who have undoubtedly passed through a previous mild and unrecognized attack of the disease and hence are of the type of true chronic carriers; as such, they are to be looked upon as particularly dan-

gerous. Others there are who but temporarily carry the organism, which soon disappears from the intestine if the source of supply, such as contaminated milk or water, is withdrawn. In these the natural resistance, whatever that may be, is such as to prevent the organism from gaining a foothold and consequently it is soon got rid of. The individual is and remains healthy, and, because of the temporary presence of the organism, is of relatively little danger to others. On the other hand, the healthy carrier may turn out to be in the incubational stage of the disease, the first symptom of which may appear in several days, or two or three weeks after the detection of the bacillus.

During convalescence from typhoid fever, presumably because of persisting lesions, the specific organism continues to be eliminated for some time. In general, however, after the tenth day following the disappearance of the fever, the typhoid bacillus disappears from the excretions of the convalescents, except from about 10 per cent. Most of the latter clear up in from three to four weeks, while others become true chronic carriers.

The chronic bacillus carrier is of especial importance, since to such, more than any other, must be ascribed the persistence of the disease in sporadic form in communities where every precaution is taken to insure a pure water-supply. This fact was most clearly established by the investigations carried on in 1902 in Alsace and Lorraine, where typhoid fever was notoriously in evidence in spite of the utmost effort to control the disease by the ordinary sanitary methods. The conclusions arrived at in the course of those studies have been verified and extended by workers in all parts of the world. The occurrence of typhoid and para-typhoid bacilli in healthy and chronic carriers can hardly be advanced as an argument against the accepted pathogenic rôle of these organisms. The lesson taught by the history of yellow fever and hog cholera might lead us to believe that the above-mentioned organisms were accidental and not causative, and that the real cause might be of an ultra-microscopic character. No experimental evidence, however, has yet been presented in support of this idea. On the contrary, all the known facts, especially the serum reactions, and above all, the remarkable results obtained in the prevention of typhoid fever by inoculation of the dead bacillus point to the etiologic significance of the typhoid germ.

The number of chronic typhoid carriers is not large, being placed by various workers at from 2 to 3 to 5 per cent.; figures which can not be considered as exact in view of the known imperfections of the methods employed. This low percentage, however, is an encouraging and redeeming feature when one considers that the excretion of the typhoid bacillus by such carriers is not always limited to weeks or months, but may continue for years and perhaps till death. This remarkable persistence of the germ is commonly considered to be due to its localization in, and adaptation to the biliary passages. Its presence in the bile and in biliary calculi is an established fact. Usually, with the bile the organism passes into the intestine to be eliminated with the discharges; less often it enters the circulation and appears in the urine. The fact that the bile is a common avenue of elimination when organisms are introduced into the blood may lead one to suppose that the bacillus is primarily localized elsewhere than in the bile bladder, and such a supposition is not without analogy. A vegetative focus within an organ not only explains the presence of the bacillus in the bile and, at times, in the urine, but also accounts for the fact that in others the

elimination is of an intermittent character.

The result of studies on the age and sex distribution of carriers indicates that children are the least, while women are most prone to this condition. The latter fact is noteworthy, since the spread of typhoid fever by carriers engaged in the handling of and preparation of food must be considered as beyond question. The figures which we have showing the relative frequency of infection through such carriers vary considerably, owing to the necessarily different conditions prevailing in different regions. Generally speaking, from 4 to 30 per cent. of the cases of typhoid fever are traceable to the chronic carrier. In localized outbreaks, such as arise in the family, the boarding house, and the like, practically every case may have this origin.

Essentially the same facts which have been developed in connection with diphtheria and typhoid fever hold true for other diseases, such as influenza, meningitis, pneumonic plague, dysentery and cholera. In the matter of cholera it may not be without interest to note that one of the most effective means employed during the past summer to prevent the introduction of cholera into this country was the systematic examination of all third-class passengers coming from infected ports for cholera carriers.

By far the most interesting and instructive example of a disease carrier is that revealed in connection with Malta fever. The cause of this disease, the *Micrococcus melitensis,* has been known for twenty-five years, but the real mode of transmission of the disease was not recognized until five years ago. It was then shown for the first time, and quite accidentally, that the goat is really a chronic carrier of the disease organism. The studies of the British Commission showed that among the many thousand of goats examined on the Island of Malta, fully 50 per cent. gave the agglutination test, while 10 per cent. were actually secreting the micrococcus in their milk. The existence of the disease among goats was wholly unexpected, but its existence forcibly taught a lesson on the importance of the chronic carrier.

The disease in the goat was so mild as to pass unnoticed. On recovery, however, the specific germ instead of disappearing became localized in the mammary gland and hence appeared in the milk. The use of this milk by man led to his infection with Malta fever. The recognition that this disease was a milk infection enabled the authorities, once and for all, to put an end to the tribute paid by the British army and navy. The simplest precaution, the boiling of the goat's milk or its avoidance, was sufficient to put an end to the scourge.

In many ways Malta fever in man presents a striking analogy to typhoid fever. In both diseases the specific organism persists in the body during convalescence and indeed after full recovery. Their continued elimination in the urine and discharges indicates a localization in some part of the body. In both infection occurs by way of the alimentary tract.

Carriers are by no means restricted to the bacterial diseases, of which but a few have been discussed. They play an even more important part in the propagation of certain protozoal infections. They constitute the natural reservoirs of virus and, as such, are chiefly responsible for the continued existence of these diseases. Thus, cattle which have recovered from Texas fever do not show, on microscopical examination of their blood, any evidence of the presence of the parasite, and yet such blood injected into a healthy animal gives rise to the typical disease. The parasite is clearly present, either in extremely small numbers, or, what is more likely, in an unrecog-

nizable form in the immune animal. The condition is analogous to that observed in the chronic carriers of typhoid fever and other bacterial diseases; it implies that a reciprocal immunity has been established, an armed truce so to speak, between the host and invader.

In trypanosomal disease chronic carriers are equally in evidence. When recovery occurs, as it does in some animals, the organism, though present in the blood, is practically unrecognizable by means other than inoculation of animals or by artificial cultivation. The culture method had revealed the presence of such trypanosomes in a large percentage of birds, and more recently the same procedure has demonstrated the existence of similar parasites in the cattle of various countries.

The disease caused by sub-microscopic or invisible organisms may show this same persistence of the infective agent long after recovery has taken place. A striking example of this fact has but recently been determined in connection with infantile paralysis where the virus has been found to persist in the naso-pharynx for many months. The existence of the chronic carrier being recognized, it is no longer surprising to learn of sudden outbreaks of this dangerous disease, apparently spontaneous in character, in a locality where no previous case was known to exist.

INVERTEBRATE CARRIERS

Important as the vertebrate carrier may be, it is quite overshadowed by the enormous importance of the invertebrate carrier. It may be asserted without fear of contradiction that the most valuable results which have been accomplished in preventive medicine in recent years have come from the recognition of the exclusive rôle played by these carriers in the transmission of many diseases. Texas fever, malaria, yellow fever, sleeping sickness, not to mention a score of other infections, find their natural transmission in the agency of insects, ticks and other sanguivorous organisms.

It has been customary for some years to speak of insects as *passive* and as *active carriers,* which terms convey certain well-defined conceptions. The passive carrier is an accidental conveyer rather than a natural host for the germ. A fly feeding upon typhoid excreta may soil its feet or proboscis and on alighting elsewhere may deposit such mechanically adhering particles. The part played by the passive carrier is merely one of indirect contamination—a purely mechanical transmission of the infective agent from one place to another. This transference of disease organisms to articles of food, or even into wounds by flying insects may lead to infection, and, in fact, it is generally recognized that certain bacterial and even protozoal diseases may thus be spread. Cholera, dysentery, tuberculosis and typhoid fever are most often mentioned in this connection.

The active carrier, on the other hand, is essentially a diseased individual and corresponds in a way to the chronic vertebrate carrier already discussed. Some, however, can be compared more correctly to the healthy carriers. The insect, tick, leech, and the like, which feed upon an infected animal may become a suitable soil for the disease organism, which either multiplies directly, or else passes through a developmental cycle in its new host. It has been supposed by some that active carriers can harbor only animal parasites, such as the pathogenic protozoa and filaria, an assumption which is quite erroneous. It is undoubtedly true that many of the known active carriers do transmit animal parasites, but that fact is not sufficient to exclude a like transmission of bacteria, nor

does it justify the assumption, so freely resorted to, that an unseen and unrecognizable germ which is transmitted by an active carrier is *ipso facto* a protozoon.

The best example of an insect bacillus carrier is seen in bubonic plague, which is spread almost wholly by the bites of fleas which come from naturally infected rats or other rodents. The plague bacillus is present in the blood of the diseased animal, usually some hours before death, and the flea which sucks up such blood into its stomach becomes infected with a variable number of the organisms. These then undergo multiplication in the digestive tube, and persist therein for four to ten to twenty-one days, depending upon the temperature, method of feeding, and perhaps on factors inherent in the flea itself. There is no evidence that the flea is affected in any way by the presence of the bacillus, which leaves the body chiefly, if not entirely, along with the feces. The contact of this excretal matter with the wound is perhaps the chief means by which infection occurs. It is clear, therefore, that the flea is not a mere passive carrier, but an actual host (a healthy carrier), even though the parasite is unable to maintain a very prolonged existence within its digestive tube.

The common rat flea is also one of the agents which brings about the transmission of a protozoal disease among rats, namely, *Trypanosoma Lewisi*. Opinions have differed as to the part played by the flea in the transmission of this infection; as to whether it was a mere passive, or a real active carrier. But recent studies have shown clearly that passive transmission, in this as in other trypanosomatic diseases, though possible, is of no special consequence; and, that the flea is a true host, an active carrier. The work of Minchin and Thomson indicates the existence of an intracellular multiplication of the parasite in the epithelial cells of the mid-gut, as a result of which the trypanosome breaks up into a number, possibly eight, daughter trypanosomes, which then become free and undergo further development and multiplication. The period of incubation is six to seven days or more, but the insect once infected remains so for an indefinite period. Regurgitation of the ripe infective form from the stomach of the flea into the wound made by the proboscis of the flea is, according to these investigators, the normal method of transmission.

Incidentally it may be stated that evidence has been presented, though it can not be said to be conclusive, which goes to show that the dog flea is responsible for the spread of the disease known as infantile kala-azar. Further investigation is needed to determine the mode of transmission of this deadly infection.

The tick family is an extremely important group as regards the transmission of disease of man and animals. The infective agent may be a bacillus, a spirochete; or a typical protozoon such as a piroplasm or a trypanosome. For example, we know from the splendid studies of Ricketts on the so-called Rocky Mountain fever, also known as "spotted" or "tick" fever, that this disease is due in all probability to a bacillus, and, furthermore, that this is invariably transmitted by the bite of a tick, or of its offspring.

The spirochetes which many workers, following the lead of Schaudinn, believed to be protozoa are now rarely classed as such even by zoologists. Instead, they have either been turned back among the spiral bacteria, or have been given a separate position, intermediate between them and the protozoa. This fact is of interest because most of the spirochetal diseases are transmitted through the agency of active carriers, such as the louse and especially ticks. The best

example of the latter type of infection is the African tick fever, which is closely identified with the ordinary relapsing fever. The active carrier in this case is the tick, *Ornithodorus moubata,* which not only can transmit the disease directly, but can also do so through its offspring. A spirochetal disease of chicken is similarly transmitted by another tick, *Argas miniatus.*

Our knowledge regarding the change which the spirochete undergoes in the tick and in the egg is by no means complete. Thus, while some workers, such as Koch, described the presence of the spiral organism in the internal organs and in the eggs, other observers have failed to demonstrate their presence. Leishman, a most careful worker, was, as a rule, unable to detect recognizable spirochetes later than the tenth day after ingestion by the ticks. Instead he observed clumps of chromatin granules which were also invariably present in the eggs, larvæ and nymphs derived from infected ticks. The spirochete origin of these granules is uncertain, especially since similar granules were found in nymphs derived from ticks fed on normal blood.

The infection of a healthy animal by a tick may occur, either by the injection of spirochetes with the salivary secretion, or by regurgitation of infective material from the gut, but neither of these modes of infection can be considered as common. Instead, it appears from the work of Leishman and of Hindle, that infection is the result of excretion of infective material from the Malpighian tubules and gut, which enters the open wound caused by the tick's bite. This contaminative wound infection is therefore similar to that already noted in connection with the flea and bubonic plague.

The Texas fever of cattle, as is well known, was the first disease in which transmission through the agency of an insect or arthropod was demonstrated. The facts presented by Smith and Kilborne twenty years ago hold to-day. Thanks to that work which served to open up the entire field of invertebrate carriers, we know that the disease is not transferred directly by the tick which has fed upon an infected animal, but indirectly through the young ticks which hatch from its eggs. The pathogenic agent in this case is a typical intracellular protozoon, the *Piroplasma.* Similar piroplasmatic diseases are met with in a variety of domestic animals and, in all such cases, transmission is effected by ticks at one stage or another of their development.

In addition to the foregoing types of organisms which are transmitted by ticks, passing mention may be made of the common trypanosome infection of cattle, already referred to, which in all probability owes its presence to this group of ectoparasites.

The recognition of mosquito-borne diseases marks one of the most important advances in modern times. Thanks to the work of Ross, Grassi and many others, we learned that malaria was transmitted solely through the bite of the mosquito. The life history of the parasite in the *Anopheles* has been traced, most completely, from the moment when it enters the stomach with the ingested blood, until it leaves the insect by way of the salivary secretion.

Barely a decade has passed since yellow fever was shown by Reed and his co-workers to be similarly transmitted, though by another genus of mosquito, the *Stegomyia.* The cause of the disease escaped their search, and for that matter is still unknown, but the practical results which followed from their work culminated in the

complete conquest of a hitherto uncontrolled scourge.

Did time permit, some consideration could be given to insect carriers, such as the body louse, which has to do with the transmission of relapsing fever and typhus fever in man, and the trypanosome infection in the common rat; the bed-bug, which has often attracted attention in connection with plague, relapsing fever and kala-azar; and to the sand-fly, which has recently been shown to be responsible for the pappataci fever, which is due, like dengue and yellow fever, to a filterable, invisible virus. It will be better to pass these by and consider briefly a vastly more important carrier, the tsetse fly.

Curiously enough, the tsetse-fly disease of South Africa was the first disease clearly recognized as insect-borne. The natives, as well as the early travelers, realized that the bite of the tsetse fly caused sickness and death of the animals thus bitten. The work of Bruce in 1894 proved this relation and at the same time demonstrated that the disease was due to a blood parasite, since named, *Tr. Brucei*. At that time Bruce was led to believe that the fly was infective for but a few hours, or at most a day or two after an infective feed. In other words, the fly was thought to be a passive carrier, simply carrying the parasite from one animal to another. This view has been definitely set aside as a result of the studies of the past two years. Mechanical transmission, especially where interrupted feeding occurs, is possible, but that this is the natural way is no longer believed. That the tsetse fly obtained its infection from the wild animals was a reasonable supposition, which was soon confirmed by special examinations. The important fact was established that the wild animal, recovering from the infection, became a chronic carrier and as such served as a natural reservoir of the virus. As long as such wild animals existed, the fly could infect itself and transmit the disease to the passing domestic animal. The introduction of rinderpest into South Africa is said to have brought about the destruction of wild animals to such an extent as to render this disease a negligible quantity in that region.

Especial interest centers about the tsetse fly because of its relation to sleeping sickness, which is caused by the *Trypanosoma gambiense*. While the fly, *Glossina palpalis*, is chiefly responsible for the spread of this disease, there is reason to believe that other species of tsetse flies can likewise serve as carriers. Attempts to eradicate the disease by removing all of the natives from certain regions have failed to accomplish the desired result. The tsetses undoubtedly obtain their infection from some other source than diseased man. This natural reservoir of the virus has not as yet been discovered, although from experiments made on antelopes it is not unlikely that these or related animals constitute the chronic carrier from which the infection is transmitted to man by the fly.

The question as to whether the tsetse fly could act as an active carrier was finally settled by Kleine (1909) and confirmed by Bruce and his co-workers. It is now known that flies which feed upon infected animals remain harmless for a period of about three weeks. After that time, however, they become infective and apparently remain so during the remainder of their life. Only a small percentage of the flies thus fed become infected. The changes which the trypanosome undergoes in the fly are but partly known. Whether this parasite has an intracellular stage, such as has been observed in fleas infected with *Tr. Lewisi*, remains to be demonstrated. It is certain, however, that *Tr. gambiense*, when it once adapts itself to the conditions in

the digestive tube of the fly, then multiplies in much the same way as it would in the culture tube. It is an interesting fact that when blood containing *Tr. brucei* is planted on a suitable culture medium an incubation period of from two to three weeks is necessary to bring about this adaptation. It is not unreasonable to believe that the changes which take place in the test-tube are not unlike those which occur within the digestive tube of the fly. The successful cultivation of *Tr. gambiense* has not as yet been realized and hence comparison of the two can not be made.

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CONCERNING NOMINA CONSERVANDA, AND A REFERENDUM TO ALL ZOOLOGISTS

The *Zoologischer Anzeiger* for January 3, 1912, publishes the result of an extensive mail vote taken among the professional zoologists of Denmark, Finland, Norway and Sweden, for and against the strict application of the law of priority in all cases, the negative vote expressing the desire that the most important and generally used names should be protected against any change on nomenclatural grounds. The vote was taken among professional zoologists, excluding anatomists, paleontologists and amateurs. Dr. Th. Mortensen, in reporting the results, comments as follows:

> The result of the vote is very striking. Of the 122 names there are two for the strict application of the priority rule in all cases, which means less than two per cent. It may perhaps not be unreasonable to conclude from this result that the number of those zoologists who swear to the strict application of the priority rule, is upon the whole very small, the great majority wishing to have the names preserved unaltered.
>
> It is to be hoped that the zoologists of other countries will follow the example given here. When this has been done and it has been definitely proved that the great majority object to the strict application of the priority rule, it may perhaps be expected that the tyranny of that notorious law, which has already done so much to damage science, will be thrown off.

The Entomological News for March, 1912, in an editorial on this subject offers to receive and print the names of any American zoologists who will send in their votes.[1] It is to be hoped that all zoologists of this country interested in the names of animals will register their votes as suggested.

Any general concurrence in the protest against the strict application of the law of priority in all cases should not be accepted as a licence for every zoologist to adopt any names that he chooses. No individual should take it upon himself to waive the rules, but in specific cases where clearly greater convenience will result from setting them aside, this should be done by such a centrally organized and authorized body as the Commission on Zoological Nomenclature. Such a body should work toward the compilation of a list of *nomina conservanda,* and the names of such a list, once adopted by the International Congress, should never be open to future change on nomenclatural grounds.

The adoption of a list of *nomina conservanda* is not without precedent. The botanists have such a list, and it appears to work well. Article 20 of the International Rules of Botanical Nomenclature reads:

> However, to avoid disadvantageous changes in the nomenclature of genera by the strict application of the rules of nomenclature, and especially of the principle of priority in starting from 1753, the rules provide a list of names which must be retained in all cases. These names are by preference those which have come into general use in the fifty years following their publication, or which have been used in monographs and important floristic works up to the year 1890. The list of these names forms an appendix to the rules of nomenclature.

The next meeting of the International Zoological Congress will occur in July, 1913, and any proposed change in the rules of nomenclature must be forwarded to the commission a year in advance of the meeting at which

[1] To Dr. P. P. Calvert, editor, 4515 Regent St., Philadelphia, Pa.